

Quantitative Evaluation of ECG Components of Workers Exposed to Carbon Disulfide

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The components of electrocardiograms (ECG) of 253 workers exposed to carbon disulfide (CDS) and those of 99 controls were quantitatively measured and evaluated. ECG of the exposed workers showed a statistically significant higher prevalence of ECG pathological changes, higher P amplitude and Macruz index, longer P duration, longer both crude and corrected Q-T intervals and R-R intervals and shorter P-R segments and QRS intervals than that of the controls. On the other hand, P-R intervals and heart rates of the two groups were not significantly different. Among both the exposed and control groups, values of P duration were significantly negatively correlated with that of P-R segment: $r = -0.216$ and -0.132 , respectively, $p > 0.05$. Values of the ECG components were not related to duration of exposure to CDS. Moreover, no significant difference was observed between duration of exposure of the exposed workers with and of those without pathological ECG changes. The method used in our study may be useful in evaluating the ECG of exposed workers before the appearance of the known pathological abnormalities.

Introduction

Carbon disulfide (CDS) is an industrial solvent used mainly in the manufacture of artificial silk and rubber. In spite of its reported toxic effects, it is still used in many countries, including Egypt. The cardiotoxic effect of CDS had been reported by many authors (1). Although CDS related electrocardiographic changes were demonstrated by some investigators (2-5), others observed the absence of such a relationship (6,7). Since electrocardiography (ECG) is a simple, quick, nonexpensive, and noninvasive technique that may be helpful in early detection of the cardiac toxic effects of CDS, more investigation may be needed to evaluate the ECG pattern of CDS-exposed workers. Accordingly, this study was initiated as a trial for quantitative evaluation of the measured components of ECG of CDS-exposed workers, which may provide an indication of the cardiac effect of CDS even in the absence of the typical known ECG abnormalities.

Subjects and Methods

The study was carried out on a random sample of 253 workers selected out of 503 workers exposed to CDS in the viscose rayon department of ESCO Factory at Bahteen (a suburb of Cairo).

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This factory is old and lacks the sufficient environmental preventive measures. The workers do not use respiratory masks. Records of the factory indicated that the level of workers' exposure to CDS ranged from 20 to 45 ppm.

A control group of 99 workers was selected from Woolltex company for spinning and weaving of wool. They were of the same age groups and occupational grade as that of the CDS exposed workers. Each member of the study and control group was interviewed about personal data, smoking habit, occupational history (which included site, nature, and duration of the present or past jobs), complaints (if any), cardiovascular-related symptoms, and past history of other diseases. This interview was followed by a short, general clinical examination. Those with past history of diabetes, liver, kidney, and rheumatic heart diseases were excluded from the study.

Electrocardiography

ECG tracing was in a quiet room in the factory clinic, using a Philips apparatus connected with the standard 12 leads (6 limb leads and 6 precordial leads). Subjects were instructed to avoid physical activity and smoking for at least 1 hr before the recording. The apparatus was regularly calibrated so that every 1 mv produced 10 mm deflexion. The speed of paper movement was 25 mm/sec.

Each electrocardiogram was examined for rate, heart rate, rhythm, p-wave duration and amplitude, P-R segment, P-R interval, QRS duration, Q-T interval, S-T segment displacement, and other pathological abnormalities (8). The criteria of these parameters are described in the footnotes of Table 1.

Table 1. Distribution percentage of the CDS-exposed workers (*n* = 253) and controls (*n* = 99) according to the value of the component of their ECG.*

ECG component						χ^2	<i>P</i>
P duration, sec	0.06–	0.08–	0.10–	≥ 0.12			
Exposed	0.80 (2)	32.4 (81)	38.80 (97)	27.74 (70)		54.3	<0.000001
Controls	2.02 (2)	74.74 (74)	14.14 (14)	9.09 (9)			
P amplitude, mm	1–	1.5–	2–	2.5–3			
Exposed	34.80 (87)	16.80 (42)	48.03 (120)	0.40 (1)		29.0	<0.00001
Controls	65.65 (65)	11.11 (11)	22.22 (22)	1 (1)			
P–R segment ^b , sec	0.02–	0.04–	0.06–	0.08–	>0.1		
Exposed	6.00 (15)	40.00 (100)	28.80 (72)	23.20 (58)	2.00 (5)	19.19	<0.0001
Controls	0 (0)	28.28 (28)	27.27 (27)	37.37 (37)	7.07 (7)		
P–R interval ^c , sec	0.12–	0.14–	0.16–	0.18–	>0.20		
Exposed	14.0 (35)	38.8 (100)	28.4 (71)	12.0 (30)	6.80 (17)	8.78	>0.05
Controls	19.19 (19)	27.27 (27)	40.4 (40)	7.07 (7)	6.06 (6)		
QRS duration ^d , sec	0.04–	0.06–	> 0.08				
Exposed	22.31 (56)	48.21 (121)	29.48 (74)			35.3	<0.000001
Controls	20.20 (20)	18.18 (18)	61.61 (61)				
Q–T interval ^e , sec	0.32–	0.34–	0.36–	0.38–	> 0.40		
Exposed	15.0 (38)	18.33 (46)	33.86 (85)	13.15 (33)	18.91 (48)	52.5	<0.0000001
Controls	44.44 (44)	9.09 (9)	41.41 (41)	3.03 (3)	2.02 (2)		
Corrected Q–T, sec	0.34–	0.36–	0.38–	0.40–	0.42–	0.44–	>0.46
Exposed	5.62 (14)	12.05 (30)	23.7 (59)	25.3 (63)	18.1 (45)	8.4 (21)	5.6 (17)
Controls	30.3 (30)	16.16 (16)	16.16 (40)	17.17 (17)	10.1 (10)	6.06 (6)	4.04 (4)
R–R interval, sec	0.5	0.7	0.9	>1.0			
Exposed	16.93 (43)	66.93 (169)	15.35 (39)	0.79 (2)		9.68	0.044
Controls	31.31 (31)	57.57 (57)	11.11 (11)	0 (0)			
Macruz index, units	1–	1.5–	2–	2.5–	> 3		
Exposed	31.2 (78)	14.40 (36)	18.40 (46)	17.20 (43)	17.81 (47)	37.3	<0.000001
Controls	63.63 (63)	8.08 (8)	18.18 (18)	5.05 (5)	5.05 (5)		

* Borders of some ECG components could not be precisely defined; numbers of cases are indicated in parentheses.

^a P-wave parameters in lead II: duration, number of small squares included between the beginning and the end of P-wave. Each small square equals 0.04 sec; amplitude, number of small squares included between the top of P-wave and the base line; each small square represents 1 mm.

^b P–R segment, number of small squares from the end of P-wave to the beginning of QRS complex; each one represents 0.04 sec.

^c P–R interval, numbers of small squares from the beginning of P-wave to the beginning of QRS complex; each small square represents 0.04 sec. This was measured in standard lead II.

^d QRS duration, number of small squares from the beginning of Q-wave to the end of S-wave; each one represents 0.04 sec.

^e Q–T interval, number of small squares from the beginning of QRS complex to the end of T-wave; each one represents 0.04 sec.

Table 2. Distribution of the electrocardiographic changes among CDS-exposed workers and controls.

Electrocardiographic changes	CDS-exposed workers n (%)	Controls n (%)
Pathological Q-wave and inverted T-wave in lead V ₁ to lead V ₆	1 (0.39)	0
Pathological Q-wave and inverted T-wave in lead III and AVF	2 (0.79)	1 (1.01)
Depressed S-T segment and inverted T-wave in leads I, AVL, and V ₆	2 (0.79)	1 (1.01)
Nonspecific ST-T changes	10 (3.95)	0
Premature ventricular contractions	4 (1.57)	1 (1.01)
A-V nodal rhythm	1 (0.39)	0
Atrial fibrillation	3 (1.18)	0
Incomplete RBBB	1 (0.39)	0
Normal ECG	229 (90.16)	96 (96.96)

The corrected Q-T interval was calculated as Q-T/R-R. The Macruz index was calculated as P duration/P-R segment (9). Statistical analysis of the results was done on IBM computer supplied with the Microstat package of statistics. Statistical tests used were Student's *t*-test, chi-square test, and Z test for two proportions.

Results

The mean ages of the CDS-exposed workers and controls were 39.37 ± 9.36 years and 41.2 ± 10.3 years, respectively. The difference is statistically insignificant, $p > 0.05$.

Smokers constituted 49 and 47% of the CDS-exposed workers and controls, respectively. The difference is statistically insignificant, $p > 0.05$. Moreover, frequency distribution of both groups as regards their systolic or diastolic blood pressure did not reveal significant differences between the two groups.

Duration of exposure of the study group ranged from 4 to 29 years (mean \pm SD = 15.4 ± 8.13 years). The prevalence of the ECG pathological changes among the exposed workers was significantly higher than among the controls (Table 2).

Frequency distribution of the two groups according to the values of the measured components of the ECG (Table 1) demonstrated statistically significant trends toward higher P amplitude and Macruz index, longer p duration, longer crude and corrected Q-T interval and R-R interval (border line significance, $p = 0.044$), and shorter P-R segment and QRS interval among the exposed group than among the controls. On the other hand, no significant difference was observed between both groups as regards P-R interval and heart rate. Among both the exposed and control groups, values of P duration was significantly negatively correlated with that of P-R segment: $r = -0.216$ and -0.132 , respectively, $p < 0.05$.

No statistically significant association or correlation could be observed between duration of exposure to CDS or smoking habit and values of the ECG components. Moreover, no significant difference was observed between duration of exposure of the exposed workers with and of those without pathological ECG changes.

Discussion

The absence of significant difference between the exposed workers and the controls in regards to age, smoking and systolic or diastolic blood pressure most probably exclude the confounding effects of such factors. The higher prevalence of pathological ECG changes among CDS-exposed workers than among controls is in agreement with data of previous reports (2-5).

The term *nonspecific S-T changes* (Table 2) is a cautious term used in the presence of S-T depression and/or T-wave inversion, which may occur in conditions other than ischemic heart disease, e.g., electrolyte imbalance, ventricular hypertrophy, metabolic disorders, acid-base balance changes, hyperventilation and drugs. These ECG changes can be considered specific when they occur during exercise or in the presence of concomitant chest pain. However, such changes should be followed up, as they may precede manifest ischemic changes (10). Elimination of this ECG abnormality from Table 2 may lead to an impression of absence of significant difference between the CDS-exposed workers and the controls. Recently, Knapikowa et al. (7) used the Minnesota code, which is a very specific code, for evaluation of the ECG of CDS-exposed workers, and they could not find significant differences between the exposed workers and the controls. Accordingly, we tried in this study to evaluate the ECG of CDS-exposed workers using the values of its measured components, which may indicate the cardiac effect of CDS before the appearance of the specific signs and symptoms of myocardial ischemia. The longer P duration and the higher P amplitude among CDS-exposed workers than among controls may point to ischemic changes. Patients with coronary heart diseases may show a broad P-wave without actual left atrial enlargement (11). The mechanisms of such prolongation may be related to delayed intra-atrial conduction (11), which may be a part of systemic affection of peripheral nerves (12).

It seems that prolongation of P duration occurs at the expense of the P-R segment. This suggestion may be documented by the observed shortening of the P-R segment of the exposed workers without significant change of the P-R intervals and by the statistically significant negative correlation between the values of P duration and P-R segment among both study and control groups. Accordingly, the use of Macruz index in indicating changes that occurred in P duration, P-R segment, and P-R interval is highly recommended because it expresses the degree of prolongation of P duration at the expense of the P-R segment. This was obvious from the markedly higher mean Macruz index of the exposed workers than that of the controls (2.0904 ± 1.054 and 1.456 ± 0.593 , respectively, $t = 7.133$, $p < 0.00001$). It should be mentioned that high Macruz index was assumed to connote impaired atrial electrogenesis even in the absence of concomitant pathological ECG changes (13).

Interestingly, the observed mean value of the Macruz index of the controls is nearly similar to that observed by Taccola et al. (13) among their control group (1.43). However, the mean value among our study group is much higher than that of Macruz index of their exposed group (1.65). This may indicate a higher

exposure effect ratio in our study group, which is most likely due to less effective environmental preventive measures and the unsatisfactory usage of personal protective devices (14).

The significance of the observed longer crude and corrected Q-T intervals among CDS-exposed workers than among the controls is difficult to establish from this study. However, changes of the Q-T interval have been reported to be associated with or precede polymorphous ventricular tachycardia (15) or even serious ventricular arrhythmias as reported in cases of organophosphorus insecticides poisoning (16).

The nonsimilarity between the results of comparisons between the exposed and control groups regarding the R-R interval and heart rate most probably are due to the fact that the two parameters were not measured exactly at the same moment. Both can be affected by many factors such as movement emotional state, respiration, etc. However, the borderline significant difference between the R-R interval of the exposed and control groups ($p = 0.044$), together with the absence of significant difference between the two groups in regard to heart rate most probably limits the value of this interval in indicating a myocardial effect of CDS-exposure. The shorter QRS duration among exposed workers than among the controls may point to acceleration of ventricular depolarization.

The absence of significant association between duration of exposure and the values of the ECG components, together with absence of significant difference between duration of exposure of the exposed workers with and of those without pathological ECG changes may indicate that the duration of exposure is not the determinant risk factor for the development of the cardiotoxic effect of CDS. This most probably supports the suggestion that CDS only manifests its coronary effect in the presence of others predisposing factors (1).

According to the results of our study, it seems that CDS had different effects on different parts of the heart. An exact explanation of such findings cannot be provided because there are no available data about studies concerning this point. However, cells of different parts of the heart have different transmembrane potentials, different thresholds, and action potentials of different shapes (17). This may lead to different responses of the different parts of the heart to CDS.

It would be interesting to investigate such changes among workers who developed pathological ECG changes. Unfortunately, this could not be demonstrated because the number of individuals in the group was too small to validate significance of the statistical analysis. It can be concluded that the method used in our study may be useful in evaluating the ECG of exposed workers before the appearance of the known pathological abnormalities.

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